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A Strategy for Research on Psychological and Social Factors in Hypertension

Richard S. Lazarus, Ph.D.

Stress-related research has thus far failed to provide an adequate understanding of hypertension and other psychosomatic ailments for three main reasons: First, there has been a continual failure to view stress as a relational phenomenon, that is, as a particular kind of transaction between person and environment. Second, there has been much confusion about the social, psychological and physiological levels of stress analysis; each is to some extent independent of the other, so that what happens at one level cannot stand for what happens at another. Third, the predominant research model has been structural and static. That is, the researcher looks at some environmental or personality characteristic, treating it as a stable property, and attempts to relate it to the risk of hypertension across persons or groups. Such an approach overlooks the key social, psychological and physiological mediating processes (e.g., social supports, cognitive appraisals, and coping) that are concurrent with and have causal significance in blood pressure elevation or change. Structural research models need to be supplemented with process-oriented ones in which the same persons are observed across various adaptational encounters and over time.

None of us needs to be reminded of our limited understanding of the etiology and mechanisms of hypertension, or of any psychosomatic illness. I use the term psychosomatic not as a particular class of illnesses, but in the cause-and-effect sense advocated by Weiss,[1] which treats all somatic illness as having potential psychosocial determinants. I plan to argue here that an important reason for our ignorance is methodological, that we need to focus in our research far more on process and less on structure, and to adopt intraindividual or ipsative strategies in which the same persons are studied repeatedly and in depth, rather than following interindividual or normative strategies.

Let me dispense at once with a principle with which, I think, many of us agree, that we are beyond the period of Koch and Pasteur in which illness was regarded as the result of an external agent, say a bacterium, and that we now recognize that illness is the result of multiple factors, including not only the virulence of the agent, but also the vulnerability of the person. I think we also agree that vulnerability can be affected by multiple physiological factors such as constitutional predisposition, temporarily lowered resistance, and so on, and multiple psychological and social factors such as psychological stress, faulty coping, social isolation or alienation, or some combination of these. The disagreements among researchers seem to center largely on which of the many physiological and psychological factors and mechanisms are most important.

When one asks about how psychological and so-

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cial factors operate, some (e.g., Cassel[2]) favor a highly general model in which stress factors are said to increase *general susceptibility* to all illnesses, while others (e.g., Graham,[3] Mason et al[4] and Lazarus[5]) suggest that there are also *specific links* between stress factors and given illnesses. Although the empirical evidence for this is as yet weak, it is quite possible that certain types of stressful relationships with the environment, particular forms of coping, or divergent emotional response qualities such as anger, anxiety, and depression, are associated with particular hormonal profiles, each with its own pull for particular tissue disturbances and illness. The dominant current viewpoint seems to favor the generality position in contrast with the specificity-centered outlook characteristic of the historically earlier pioneers in psychosomatic medicine (see Alexander and Selesnick,[6] and Lipowski[7]).

THE STRUCTURAL RESEARCH MODEL

Why should it be true that previous psychosomatic research has not led us to the holy grail of understanding? There are two main reasons: First, there has been a failure to understand the *concept of stress* itself at each of three levels of analysis, the social, psychological, and physiological. The key defect is the continuing tendency to separate artificially the two major classes of interacting causal antecedents of the stress response, namely, the external environment and characteristics of the person. Again and again writers seem to be saying that stress as an agent (stressor) is either out there in the social or physical environment, or within the person as an intrapsychic defect. As my colleagues and I[8,9] have been at pains to point out, however, stress is a special kind of *transaction* or relationship between a particular kind of system (a person, social unit, or tissue structure) and a particular kind of environment. Although all levels of analysis are equally important, stress has quite different meanings at each.

At the psychological level, with which I am especially concerned, stress depends on the ways an individual cognitively *appraises* a transaction and *copes* with it. The individual may view it as threatening, challenging or benign, and cope by direct action to change things or by intrapsychic processes such as denial, detachment or by trying to avoid thinking about the negative aspects. These mediational processes profoundly alter the tissue state of the person and hence his or her prospects of developing a disorder such as hypertension. Until individual differences in the processes of appraisal and coping are understood and brought into the equation, the role of psychological stress in hypertension and other somatic disturbances cannot possibly be understood, and research will continue to fail to specify adequately the crucial personality and situational variables which are causal because they affect the way a person appraises his or her relationships and copes.

Second, if one believes the dictum that "It's what you do hour by hour and day by day that largely determines the state of your health, when you get sick, what you get sick with, and perhaps when you die," then one must realize that the traditional way hypertension is studied will fail because the typical research method is almost exclusively static or *structural* rather than *process*-oriented. The most characteristic research method is to look at some environmental characteristic such as job loss, a high-pressure work situation, or some personality characteristic or trait, to determine whether the risk of hypertension is greater than for those working under non-stressful circumstances or not manifesting the trait.

Aside from anecdotal clinical observation, large samples are typically used in such research, and assessments are usually made once rather than repeatedly. They are designed to measure presumably stable psychological tendencies or chronic environmental conditions. Such observations are, perforce, incomplete because they ignore the multileveled and variable transactional processes taking place from setting to setting, or moment to moment, that *mediate* whatever statistical relationship between cause and effect variables might be suggested by the typical normative study. The individual psychodynamic processes and their concurrent physiological correlates and mechanisms are implied rather than directly examined, yet it is just these processes that make it possible for any characteristic, either personal or environmental, to be expressed in a somatic disturbance such as hypertension.

The emphasis on structure has been criticized by others, too. For example, Stahl and his colleagues[10] express their dissatisfaction in the following way:

> "The models used by both disciplines (epidemiology and behavioral science) can be characterized as structural models. In such a

model, sociological and psychological variables are directly related to the disease entity without considering the influence of intervening processes which are directed to some goals and which serve to make the somatic structure a dynamic model. For example, social epidemiologists tend to test the interactive process between the structure (social class) and the behavior (hypertension). Using a more psychologically oriented example, the association between hostility (a personality characteristic or structure) and hypertension is postulated without testing directly the conceptual linkage between structure and behavior."

The structural research model pushes us toward three kinds of incomplete alternative statements about the etiology of hypertension: (1) certain types of people (with particular personalities and/or constitutional predispositions) are more likely than others to develop hypertension; (2) any person is more likely to show hypertension under certain kinds of environmental settings (e.g., pressured jobs, such as those of air traffic controllers); (3) there is some form of interaction between statements (1) and (2) such that both sets of factors are necessary to produce hypertension, or that both factors together increase the risk additively or in accordance with some other mathematical function.

THE PROCESS-ORIENTED RESEARCH MODEL

What is missing from the typical structurally oriented, interindividual or normative pattern of research is the study of actual transactional processes taking place between a given type of person (defined personalistically or in terms of physical constitution) and given types of environmental settings. Crucial to understanding hypertension is the comparison of those psychological processes that lead to raised blood pressure and those that do not. We must ask what it is that the hypertensive is doing when his or her blood pressure is up and when it is down, and what the normotensive is doing to produce similar variations. The essential difference between the two populations is that the hypertensive presumably has repeated or chronic transactions capable of raising blood pressure while the normotensive has few, and/or a greater constitutional disposition to react under stress in the sphere of blood pressure than in some other organ system. To evaluate both possibilities and flesh them out requires that adaptational transactions be studied over time and across many types of environmental settings. The crux of the matter is to study the ebb and flow of blood pressure *concurrently*[11] with psychological processes such as cognitive appraisal and coping, so that these processes, the psychological and tissue, can be tied to each other.

A recent discussion in the *Journal of Human Stress*[12] concerning hypertension will point up the distinction between structural and process-oriented research. In one comment, Rose refers to persons who show significant elevations in blood pressure in a variety of situations, implying the presence of a stable hypertensive trait or symptom pattern. Similarly, in referring to evidence that air traffic controllers commonly show hypertension, Rose implies a structural property of a specific work setting. On the other hand, process is implicated when, in that discussion, Jenkins asks the following question about the case under consideration: "In terms of his current life situation, what are the psychological concomitants of his sweaty palms, high pulse, and elevated blood pressure? Do frightening ideas go through his mind? Does he perhaps ruminate over the means by which he originally raised his blood pressure?" Finally, when Rose cites air traffic controllers who show very large changes in blood pressure at different times during their work day, the process-oriented question about what such men are doing, thinking and feeling while their blood pressures are either high or low, cries out to be answered.

To do this requires a very different research strategy than is typically used. We might start with a given type of group, say, those who have the clinical symptoms of hypertension, and compare it with a group of normotensives. However, the strategy must be intraindividual as well as interindividual: The same persons must be examined across a variety of settings and transactions. Elsewhere, this style of research has been referred to as ipsative-normative,[8,13,14] ipsative meaning the intra-individual study of various facets of the same individual or given individuals seen in different contexts, and normative meaning interindividual study of one or several properties in the population at large or in a particular group. An excellent treatment of this issue has been made recently by Marceil.[15]

Since much of this type of research effort is taken up in studying the same persons in a variety of contexts, and over and over again through time, process-oriented research cannot afford the large numbers of cases characteristic of most epidemiological research. The trade-off is basically simple to understand. At one extreme, in strictly normative or interindividual research we are looking at a single property or a few properties, say, of personality or of the environment, and seeking to evaluate whether or not that property affects health outcome, say, the risk of hypertension. Since the amount of variance accounted for by a single variable or a few variables is usually small, it pays then to use a large number of cases, and we are likely to end up with a relationship that is statistically significant but that may be minor in importance — that is, it leaves much variance unaccounted for. At the other extreme, in strictly idiographic research, we settle for a single clinical case as a source of hypotheses, and we can carefully study multiple psychological processes and facets of personality. The ipsative-normative research I am recommending is somewhere in between: that is, we can study many things about a relatively small number of persons, or we can examine them under many environmental contexts, and over time.

For example, my colleagues and I are presently assessing what is happening to subjects over the course of a year, sampling such events once a month, and observing multiple social and psychological processes taking place each month. We are looking at major life changes, daily hassles and positive events, patterns of emotion sampled regularly, and patterns of coping as employed in specific stressful transactions. We want to relate these events and mediating processes to outcomes such as health/illness, morale, and social functioning. As one might imagine, looking as we are at ongoing processes, the little dramas of daily life, as it were, requires quite different measurement strategies than assessing stable traits or stable situational pressures. And to pay sufficient attention to such mulitple facets and processes within the same person, the large samples so useful in epidemiologoical research must be sacrificed. Biostatisticians might well look askance at the small samples necessitated in such research, but we believe any disadvantage is well compensated for by the rich opportunities ipsative-normative-style research provides to view the person and the important, ongoing psychophysiological processes, in many lights and in many divergent environmental settings. Above all, this research strategy allows us to observe the flux and change characteristic of human adaptation.

Applied to the problem of hypertension, it should be noted that even adaptational outcome variables can be viewed in process terms, since these wax and wane or change over time as a function of a variety of antecedent and mediating process variables. In any event, process-oriented research permits us to address many questions related to etiology and mechanisms that have been hitherto refractory to research.

It might be argued that process-oriented research cannot study mediating processes without someone first suggesting or establishing which variables are, indeed, probable or potential mediators of the relationship between stress and somatic illness. Therefore, process-oriented research, short of being fishing expeditions swamped with a mass of irrelevant material, is ultimately dependent on structural research that identifies the mediating variables. This argument has some validity. It is correct to say that process-oriented research needs some guides about the potential mediators that might be profitable to study. Although such guides can come from structural research models if we are lucky, they also come from sociodynamic and psychodynamic theory. Therefore, a more modest statement of my position would be that process-oriented research is a necessary feature of any attempt to link social and psychological factors with illness outcomes. Both kinds of research, structural and process-oriented, are essential to adequate understanding, and since the latter has been underemphasized of late, I feel secure in attempting to argue passionately and one-sidely about the need to redress the balance.

ARE WE READY FOR A "PARADIGM SHIFT"?

Professor Syme, my colleague on the Berkeley campus, has been flirting with the same methodological position, though I am not sure he has yet embraced it as wholeheartedly as I. In one recent paper, Syme[16] draws on the suggestion by Ostfeld[17] that there is substantial epidemiological evidence linking hypertension to potentially harmful environmental contexts characterized by a situation with an uncertain outcome, in which fight or flight is inappropriate, and in which the person must maintain constant vigilance. The evidence Syme cites includes research on job loss,[18] the pressures faced by air traffic controllers,[19] men living in crowded penal institutions,[20] and persons living in stressful urban environments.[21]

In a related paper, however, Syme[22] appears to recognize the limitations of such structurally oriented, large-scale normative research, considering the findings as suggestive rather than definitive because they involve merely correlations between a set of environmental circumstances and increased risk of hypertension, without the psychophysiological mechanisms mediating the relationship being identified. Acknowledging the need to identify the psychophysiological mechanisms, Syme cites research by Obrist et al [23] who report that subjects withdraw when they sense they cannot control their environment, a mode of coping that permits vagal rather than adrenergic regulation of heart rate and blood pressure.

The basic transaction in hypertension is said to be one in which the person is psychologically geared to cope actively and so mobilizes rather than withdraws. Active or involved coping should produce somatic mobilization; defensive coping such as denial or detached avoidance should not. Notice that it is not a particular type of environmental stressor about which I am speaking now, for example, high population density, job loss, or whatever, but a far more complex set of mediating psychological factors and processes, including the way a person appraises the transaction and his or her choice of coping strategy. One cannot help but think here of an earlier finding by Weiner, Singer and Reiser[24] that hypertensives, previously identified as such clinically, tended to defend themselves against a potentially emotional clinical interview by insulating themselves, that is, by becoming detached or uninvolved. Paradoxically, hypertensives showed a lower blood pressure response to the research interview than normotensives did. Although we might be wary about the fact that hypertensives, who are by implication generally mobilized, used this mechanism, the finding seems to highlight what I have been saying about the importance of mediating psychological processes such as appraisal and coping (see also Sapira et al[25] for additional, confirming observations).

To my mind, Syme's recognition that the dynamics of the transaction must be studied in order to learn what is happening to the hypertensive is an important methodological step forward. Obrist and his colleagues have made, in effect, a process analysis in the laboratory in that they studied the conditions under which blood pressure will or will not rise. The main trouble, however, is that laboratory research is only one of the number of ways to get at such dynamics, and, in my view, not the best method at our particular stage of understanding for a number of reasons. Most important, it cannot tell us about the ecological conditions under which hypertensives and normotensives live, the forms of coping such persons employ, and the impact of all this on their blood pressure under the natural conditions of their lives.

An effort to assess the psychological, social and physiological processes underlying hypertension and other psychosomatic ailments requires well-designed field studies of the same persons, ideally in their natural habitats, and the development of new tools of measurement for describing such processes. We need to supplement large-scale epidemiological studies with small-scale but more intensive efforts directed at various target persons and groups. We must study such persons and groups in daily life situations, not just once or twice statically, but repeatedly over time, and thoroughly enough to obtain a valid in-depth picture of the relevant social, psychological and somatic processes that occur as part of that person's stressful transaction with the environment.

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INDEX TERMS

hypertension, stress, coping, intraindividual (or ipsative) research design, psychosomatic medicine, process measurement.

REFERENCES

1. Weiss, J. "The Current State of the Concept of Psychosomatic Disorder," *Psychosomatic Medicine*, Z. J. Lipowski, D. F. Lipsitt, and P. C. Whybrow, eds., pp. 162-171. Oxford University Press, New York, 1977.
2. Cassel, J. "The Contribution of the Social Environment to Host Resistance," *Am. J. Epidemiol.*, Vol. 104, 1976, pp. 107-123.
3. Graham, D. T. "Some Research on Psychophysiological Specificity and Its Relation to Psychosomatic Disease," *Physiological Correlates of Psychological Disorder*. R. Roessler and N. S. Greenfield, eds. University of Wisconsin Press, Madison, 1970.

4. Mason, J. W., J. T. Maher, L. H. Hartley, E. Mougey, M. J. Perlow, and L. G. Jones. "Selectivity of Corticosteroid and Catecholamine Response to Various Natural Stimuli," *Psychopathology of Human Adaptation*, G. Serban, ed., pp. 147-171. Plenum Press, New York, 1976.
5. Lazarus, R. S. "Psychological Stress and Coping in Adaptation and Illness," *Psychosomatic Medicine: Current Trends and Clinical Applications*. Z. J. Lipowski, D. R. Lipsitt, and P.C. Whybrow, eds., pp. 14-26. Oxford University Press, New York, 1977.
6. Alexander, F. G., and S. T. Selesnick. *The History of Psychiatry*, ch. 24. Harper & Row, New York, 1966.
7. Lipowski, Z. J. "Psychosomatic Medicine in the Seventies: An Overview," *Am. J. Psychiatry*, Vol. 134, 1977, pp. 233-244.
8. Lazarus, R. S., and J. B. Cohen. "Theory and Method in the Study of Stress and Coping in Aging Individuals," paper given at the Fifth W.H.O. Conference on Society, Stress and Disease: Aging and Old Age, Stockholm, Sweden, June 14-19, 1976.
9. ________ "Environmental Stress," *Human Behavior and the Environment: Current Theory and Research*, I. Altman and J. F. Wohlwill, eds. Plenum Press, New York, in press.
10. Stahl, S. M., C. E. Grim, S. Donald, and H. J. Neikirk. "A Model for the Social Sciences and Medicine: The Case for Hypertension," *Soc. Sci. Med.*, Vol. 9, 1975, pp. 31-38.
11. Luborsky, L., J. P. Docherty, and S. Penick. "Onset Conditions for Psychosomatic Symptoms: A Comparative Review of Immediate Observation with Retrospective Research," *Psychosom. Med.*, Vol. 35, 1973, pp. 187-204.
12. "Can Hypertension Be Induced by Stress? A Case Discussion," *J. Hum. Stress*, Vol. 3, 1977, pp. 4-11.
13. Lazarus, R. S. and R. Launier. "Stress-Related Transactions Between Person and Environment," *Internal and External Determinants of Behavior*, L. A. Pervin and M. Lewis, eds. Plenum Press, New York, in press.
14. Broverman, D. M. "Normative and Ipsative Measurement in Psychology," *Psychol. Rev.*, Vol. 4, 1962, pp. 295-305.
15. Marceil, J. C. "Implicit Dimensions of Idiography and Nomothesis: A Reformulation," *Am. Psychol.*, Vol. 32, 1977, pp. 1046-1055.
16. Syme, S. L. "'Benefit-to-Risk' Ratio Involved in the Drug Treatment of Mild Hypertension: Social and Psychological Considerations," paper read at New York Academy of Sciences Conference on Mild Hypertension, New York, N.Y., February 9-11, 1977.
17. Ostfeld, A. M. "The Interaction of Biological and Social Variables in Cardiovascular Disease, *Milbank Mem. Fund Q.*, Vol. 45, 1967, pp. 13-18.
18. Kasl, S. V., and S. Cobb. "Blood Pressure Changes in Men Undergoing Job Loss: A Preliminary Report," *Psychosom. Med.*, Vol. 32, 1970, pp. 13-18.
19. Cobb, S., and R. M. Rose. "Hypertension, Peptic Ulcer, and Diabetes in Air Traffic Controllers," *J. A. M. A.*, Vol. 224, 1973, pp. 489-492.
20. D'Atri, D. A., and A. M. Ostfeld. "Crowding: Its Effects on the Elevation of Blood Pressure in a Prison Setting," *Prev. Med.*, Vol. 4, 1975, pp. 550-566.
21. Harburg, E., W. J. Schull, J. C. Erfurt, and M. A. Schork. "A Family Set Method for Estimating Heredity and Stress. I. A Pilot Survey of Blood Pressure Among Negroes in High and Low Stress Areas, Detroit, 1966-1967," *J. Chronic Dis.*, Vol. 23, 1970, pp. 69-81.
22. Syme, S. L. "Psychosocial Determinants of Hypertension," paper presented at Hahnemann College's Fifth International Symposium on Hypertension, San Juan, Puerto Rico, January 9-12, 1977.
23. Obrist, P.A., A. W. Langer, A. Griginolo, J. R. Stutterer, K.C. Light, and J. A. McCubbin. "Blood Pressure Control Mechanisms and Stress: Implications for the Etiology of Hypertension," paper read at Hahnemann College's Fifth International Symposium on Hypertension, San Juan, Puerto Rico, January 9-12, 1977.
24. Weiner, H., M. T. Singer, and M. F. Reiser. "Cardiovascular Responses and Their Psychological Correlates. I. A Study in Healthy Young Adults and Patients with Peptic Ulcer and Hypertension," *Psychosom. Med.*, Vol. 24, 1962, pp. 477-498.
25. Sapira, J. D., E. T. Scheib, R. Moriarty, and A. P. Shapiro. Differences in Perception Between Hypertensive and Normotensive Populations," *Psychosom. Med.*, Vol. 33, 1971, pp. 239-250.